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Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health

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Running Title: Cardiovascular Health and Ultrafine Particles

Key words: epidemiology, toxic air pollutants, diesel, cardiovascular diseases, particle size, oxidative stress, cytokines.

Abbreviations:

BP : blood pressure

CAP: concentrated ambient particles

CIMT: carotid intima-media thickness

CI: confidence interval

CHD: coronary heart disease

CHF: congestive heart failure

COPD: chronic obstructive pulmonary disease

DEP: Diesel exhaust particles

EC: elemental carbon

ETS: environmental tobacco smoke

HR: heart rate

HRV: heart rate variability

IL-1β: interleukin 1beta

IL-6: interleukin 6

$NC_{0.01\text{-}0.1}$: number concentrations of ultrafine mode particles 0.01 to 0.1 μm in diameter

$NC_{0.1\text{-}1}$: number concentrations of accumulation mode particles 0.1 to 1.0 μm in diameter

NFκB: nuclear transcription factor-κB

NMMAPS: National Morbidity, Mortality and Air Pollution study

Odds ratio: OR

PAH: polycyclic aromatic hydrocarbon

PM: particulate matter

$PM_{2.5}$: particulate matter < 2.5 μm in aerodynamic diameter

PM_{10}: particulate matter < 10 μm in aerodynamic diameter

PN: particle number

ROS: reactive oxygen species

RR: relative risk

TNF-α: tumor necrosis factor-α

TSP: total suspended particulates (PM approximately < 50 μm in diameter)

UFP: ultrafine particulate matter, < 0.1 μm in aerodynamic diameter

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OUTLINE OF MANUSCRIPT SECTION HEADERS:

ABSTRACT

Numerous epidemiologic time series studies have shown generally consistent associations of cardiovascular hospital admissions and mortality with outdoor air pollution, particularly mass concentrations of particulate matter (PM) < 2.5 or < 10 μm in diameter ($PM_{2.5}$, PM_{10}). Panel studies with repeated measures have supported the time series results showing associations between PM and risk of cardiac ischemia and arrhythmias, increased blood pressure, decreased heart rate variability, and increased circulating markers of inflammation and thrombosis. The causal components driving the PM associations remain to be identified. Epidemiologic data using pollutant gases and particle characteristics such as particle number concentration and elemental carbon has provided indirect evidence that products of fossil fuel combustion are important. Ultrafine particles < 0.1 μm (UFP) dominate particle number concentrations and surface area, and are therefore capable of carrying large concentrations of adsorbed or condensed toxic air pollutants. It is likely that redox active components in UFP from fossil fuel combustion reach cardiovascular target sites. High UFP exposures may lead to systemic inflammation through oxidative stress responses to reactive oxygen species, and thereby promote the progression of atherosclerosis and precipitate acute cardiovascular responses ranging from increased blood pressure to myocardial infarction. The next steps in epidemiologic research are to identify more clearly the putative PM casual components and size fractions linked to their sources. To advance this, we discuss in a companion paper (Sioutas et al. in press) the need for and methods of UFP exposure assessment.